4- THERMOREGULATION -II

Prof. Sherif W. Mansour

Physiology dpt., Mutah School of medicine

CONTROL OF BODY TEMPERATURE

The thermoregulatory system is composed of:

-Sensory receptors (thermoreceptors)

- Central integrator (center)

- Effector organs

I- Thermoreceptors:

A) Peripheral thermoreceptors:

Site : -Skin contain both cold and warmth receptors with more cold receptors.

-Deep body temperature receptors are present in abdominal viscera and spinal cord to detect body core temperature

Pathway : the peripheral thermoreceptors discharge impulses via the lateral spinothalamic tract to the thalamus and somatosensory cortex with collateral from the thalamus pass to activate the heat regulatory center in hypothalamus.

B) Central thermoreceptors:

- The **anterior hypothalamus** and the preoptic area contain large number of heat sensitive neurons and cold sensitive neurons.

- These receptors are sensitive to core temperature (brain & blood temp.).

II-Thermoregulatory center (thermostat):

- It is present in the hypothalamus.

- It receives impulses from the thermoreceptors and compares it with its specific standard reference temperature (**set – point**) which almost 37.1 C° body core temperature.

- if body temp. higher than set point → stimulation of anterior hypothalamus → heat loss.

- if body temp. less than set point → stimulation of posterior hypothalamus → ↑ heat production & ↓ heat loss.

III-The effectors organs :

-skin (blood vessels and sweat gland)

-Skeletal muscle

- Endocrine glands

Effects of exposure to cold environment

Exposure to cold stimulates the **posterior nuclei of the hypothalamus** which regulate the heat balance by:

A) Decrease in heat loss:

•Vasoconstriction of skin blood vessels.

The posterior hypothalamus stimulates the vasoconstrictor center in the medulla → sympathetic adrenergic fibers → v.c. So the skin becomes cold as the weather. With no heat loss occurs. Also, sympathetic stimulation to skin causes piloerection which act as insulator layer around skin.(little effect in human).

•Counter-current heat exchanger:

- as vasoconstriction of cutaneous blood vessels directs blood to deep veins which run parallel to the arteries.

-Heat is conducted from the warm arterial bl. to the cold venous blood → warm venous blood return to the heart and cold arterial bl. to skin.

•Behavioral responses:

-Putting on heavy clothes.

-Curling the body to decrease surface area.

-Erection of hair "**horripilation**" as an insulator for cold (sympathetic effect).

B) Increase in heat production:

•**Shivering: I**t is involuntary rhythmic contractions of the skeletal muscle to produce large amount of heat. Its center is present in **posterior hypothalamus** in area called the **primary motor center for shivering** (this area is normally inhibited by impulses from heat center in the anterior hypothalamic preoptic area), this center stimulates the extrapyramidal tracts → AHCs to increase ms. tone to high level → shivering.

-shivering can be prevented by curare (neuromuscular blocker).

•**Hormonal thermogenesis:**

a) Adrenaline: Hypoth. stimulates the adrenaline secreting center in M.O which stimulate suprarenal medulla → adrenaline → Increase metabolic rate.

- Cutaneous vasoconstriction.
- Stimulate glycogenolysis.
- Stimulate lipolysis (of depot fat which is rich in children).

b) Thyroxin:

Hypoth. → Thyrotropin releasing hormone → stimulate anterior pituitary gl. to secrete thyroid stimulating hormone → stimulate thyroid gland to secrete thyroxin hormone → ↑ BMR slowly (in **cold** climate there is high incidence of **toxic goiters**).

c) Cortisol:

Hypoth. → corticotrophin releasing factor → stimulates Anterior Pituitary → ACTH → stimulate adrenal cortex → ↑ cortisol → ↑ blood glucose and metabolic rate.

•**Behavioral responses:** Increase **appetite** to increase **Specific Dynamic Action** that lead to more liberation of heat.

Effect of exposure to heat

Exposure to heat stimulates the anterior nuclei of hypothalamus which regulate body heat balance by:

A) Decrease heat gain:

By strong inhibition of mechanisms that cause heat production and by behavioral responses as apathy (decreased activity) and anorexia (decrease appetite).

B) Increased heat loss:

***Vasodilatation of skin blood vessels:**

It is caused by: - Inhibition of the sympathetic centers in post. Hypoth. □ VD □□ heat transfer to skin then lost to surrounding.

-Direct effect of heat on skin.

-Release of bradykinin from sweat glands.

***Sweating:** Sweat is hypotonic secretion of Nacl. Its center is the **preoptic nuclei** in the anterior hypothalamus.

-The nerve supply of **eccrine sweat glands** are supplied by sympathetic cholinergic fibers (blocked by atropine).

-Sweat secretion: is an **active process** in which the acini secrete **isotonic** sweat (**primary sweat**) as plasma, but NaCl is gradually reabsorbed by the ducts so the sweat becomes **hypotonic** (**secondary sweat**) (this change depends on the rate of sweating as increase rate of sweating doesn't give time of modification of the primary sweat, also depends on aldosterone level which increase NaCl reabsorption).

- Cooling effect of sweat:

-Each 1 ml evaporated sweat removes 0.6 K Cal.

-Sweat start at environmental temp of 32 C°

-Dribbling alone without evaporation → no loss .

-**Acclimatization of Sweating**:

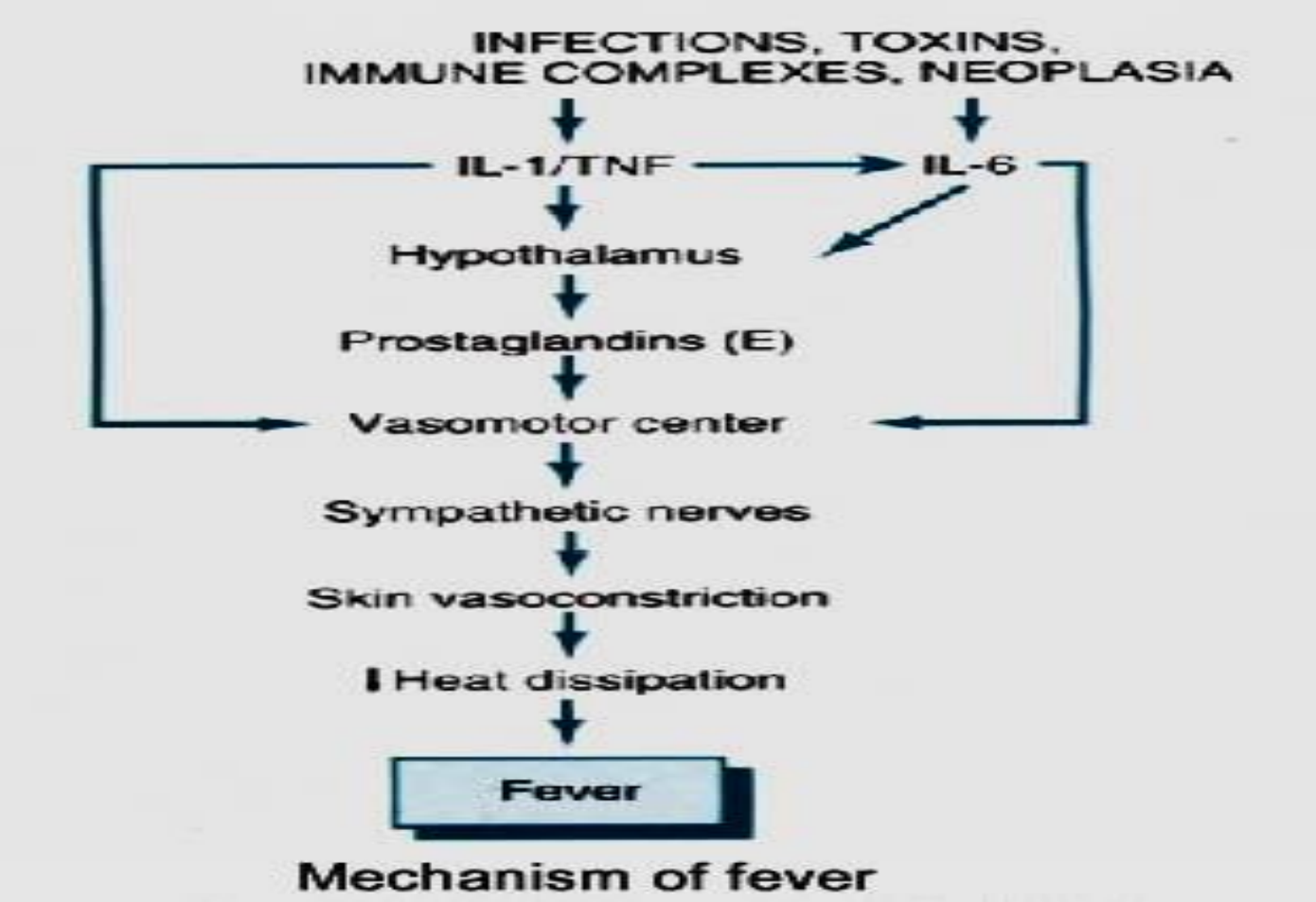
In acute exposure to hot wheather: A person sweat 700 ml/h + loss of 15-30 gm Nacl/day. After exposure to hot weather for 6 weeks, he sweat 2000 ml/h and Nacl loss 3-5 gm/day (due to increase sweating capability of the sweat glands also due to ↑ aldosterone secretion by the associated decrease in NaCl level in the body)

N.B.: **Cold sweat** is emotional sweating even with cold and vasoconstriction.

Disorders of temperature regulation

•**Fever (pyrexia):**

- It is hyperthermia caused by resetting of the set-point of the hypothalamus to a higher level.



Mechanism of fever

Mechanism of fever:

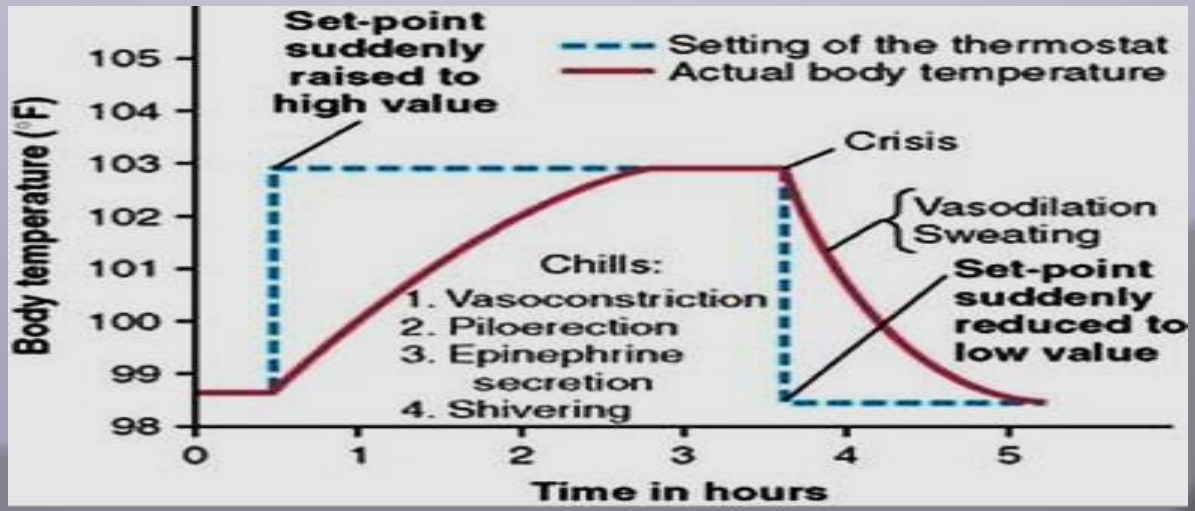
1-Toxins of bacteria + degenerated tissue → **exogenous pyrogens** which act on the monocytes and macrophages which release interleukin and tumor necrosis factor which are called endogenous pyrogens.

2-The **endogenous pyrogens** (IL-1) reach the hypothalamic thermosensitive neurons cause fever within ten minutes by formation of prostaglandin E2 (PGE2) which activates resetting of the central thermostat (↑ set-point) by synthesis of cAMP.

3-Because body temp. is still less than the set-point, the person has false feeling of cold and mechanisms of elevation of body temperature occur → v.c with cold skin and **shivers (chills**) which continue till the body temp. is elevated to the level of set-point → fever.

4 -**The crisis (Flush):**

If the factor that causes fever is removed (treated), the set-point returns to the normal level and the body temp. is still more than the set-point → sensation of hotness →↑ mechanism of heat loss → VD (flushed skin) and intense sweating → return the body temp. to normal level.



Control of fever:

- PGE2 has a negative feed back on interleukin I.
- ↑ interleukin I → down regulation of its receptors.
- **Glucocorticoids** as cortisol →↓ interleukin I.
- **Aspirin** as antipyretic drugs →↓ synthesis of PGE2 from arachidonic acid →↓ set point level →↑ heat loss by sweating (aspirin doesn't lower body temperature of a normal person because normal person doesn't have any interleukin-1). Also, aspirin stimulates the heat losing center.

Heat stroke

Cause: this occurs due to exposure to hot humid weather or to high fever.

Mechanism:↑ body temp. → excessive sweating → dehydration and salt loss → depression of heat regulating center →↓ sweating & dry hot skin →↑ body temp. at 43oC → irreversible denaturation of tissue proteins → depression of the center (vicious circle) → death.

Clinical picture: Dizziness, loss of fluids and sweat may lead to circulatory shock also, degeneration of body tissue may occur.

Treated by:

-Immediate cooling of the body by immersion in ice cold water.

-Sponge with alcohol.

-Antipyretic drugs as aspirin.

Sun stroke

Beside sweating and dehydration damage of brain tissue by direct sun rays → severe fever, it is treated by immersion in ice bath and drinking saline.

-Physiological changes associated with **hyperthermia**:

1- Central Nervous system:

- At first, hyperthermia stimulates the CNS leading to tremors and convulsions. But at body temperature above 41C° malfunction of CNS occur leading to loss of reflexes and coma.

2- Cardiovascular system:

-Increase heart rate by 10 beats/min for each 1C° increase in body temperature due to direct stimulation of SAN or cardio-accelerator center.

-Increase cardiac output due to vasodilatation of peripheral arterioles with increase in venous return.

-Increase systolic blood pressure (by increase C.O.P) and decrease diastolic blood pressure (by peripheral vasodilatation) but on exposure to **hot humid atmosphere**, excessive sweating and vasodilatation will lead to dehydration and hypotension, a condition known as "**heat exhaustion**".

3- Respiration:

- Increased respiratory rate by stimulation of the central and peripheral chemoreceptors.

Malignant hyperpyrexia

It is a dangerous complication of **general anesthesia** occurring in individuals with an underlying disease of muscle.

The essential clinical features of the syndrome are a drastic and sustained rise in body temperature, metabolic acidosis, and widespread muscular rigidity.

This is caused by a massive and sudden release of **calcium** into the myoplasm from the calcium-storing membranes in the muscle cell when exposed to general anesthetic agents.

Susceptible individuals should be given **local, regional, or spinal anesthesia** if an operation is needed. If this form of anesthesia is unsuitable, **barbiturates** such as thiopentone, **tranquilizers** such as diazepam, **narcotics** such as Pantopon, and **neuroanaleptics** such as fentanyl, nitrous oxide, d-tubocurarine, and althesin appear to be safe.

By far the most important aspect of treatment is **prophylaxis**. Early diagnosis and immediate cessation of the offending anesthetic agents are the most important factors in trying to reduce the very high mortality of the syndrome.

Hypothermia

It is a drop of body temperature to low level with slow metabolic and physiologic processes (respiration, heart rate,..

Causes:

1. Exposure of the body: to extreme cold water (ice water) for 20 minutes →↓ body temp. to 35C° → heart stop. The ability of the hypothalamus to regulate body temperature is greatly **impaired** with sleepiness and even coma occur.

2. Frost-bite: exposure of the body to extreme cold weather → freezing in lobes of ears and digits of hands & feet (frost – bite) may lead to gangrene and loss of these areas.

3. Artificial hypothermia: by strong sedative which depress heat regulating centers or cooling the patient with ice. This is used to stop the heart artificially for many minutes during cardiac surgery.

Thank You